APPENDIX C

HUMAN HEALTH

Lake Michigan Lakewide Management Plan: Human Health Appendix

April 2000

.Lake Michigan Lakewide Management Plan (LaMP): Human Health Appendix

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Lake Michigan Lakewide Management Plan (LaMP): Human Health Appendix

1.0 Introduction

1.1 Great Lakes Water Quality Agreement and the Lakewide Management Plan

The purpose of the Great Lakes Water Quality Agreement of 1978, a binational agreement between the United States and Canada, "is to restore and maintain the chemical, physical, and biological integrity of the waters of the Great Lakes Basin Ecosystem" (IJC 1994). The Agreement calls for the establishment of Lakewide Management Plans (LaMPs) for open lake waters. These plans are ". . .designed to reduce loadings of Critical Pollutants in order to restore beneficial uses" (IJC 1994), in the form of fish and drinking water consumption, and recreational water use. In addition, the LaMPs are required to define [describe] the threat to human health from Great Lakes Critical Pollutants (substances that may cause impairment of beneficial uses). The International Joint Commission (IJC), which assists in the implementation of the Agreement, has identified 11 Great Lakes Critical Pollutants (IJC 1998). They are alkylated lead, benzo[a]pyrene, DDT [1,1,1-trichloro-2,2-bis(p-chlorophenyl)ethane] and metabolites, dieldrin, dioxins, furans, hexachlorobenzene, methylmercury, mirex, PCBs (polychlorinated biphenyls), and toxaphene (Table 1). In addition, the broader Agreement calls for recreational waters to be substantially free from bacteria, fungi, and viruses, and also calls for control of contaminated groundwater (drinking water).

1.2 Health Objectives of the Lake Michigan LaMP Human Health Appendix

The health objectives are to support the human health requirements of the Great Lakes Water Quality Agreement by 1) defining (describing) the potential adverse human health effects arising from exposure to Critical Pollutants and other contaminants found in Lake Michigan and other connecting Great Lakes and waterways, 2) examining societal responses (i.e., implementation strategies), such as health advisories for fish consumption to minimize exposure to contaminants found in fish from Lake Michigan and other Great Lakes, and 3) making

recommendations for continued research in areas such as chemical mixtures effect and endocrine disruptors (Table 2).

Table 1: International Joint Commission's Critical Pollutants

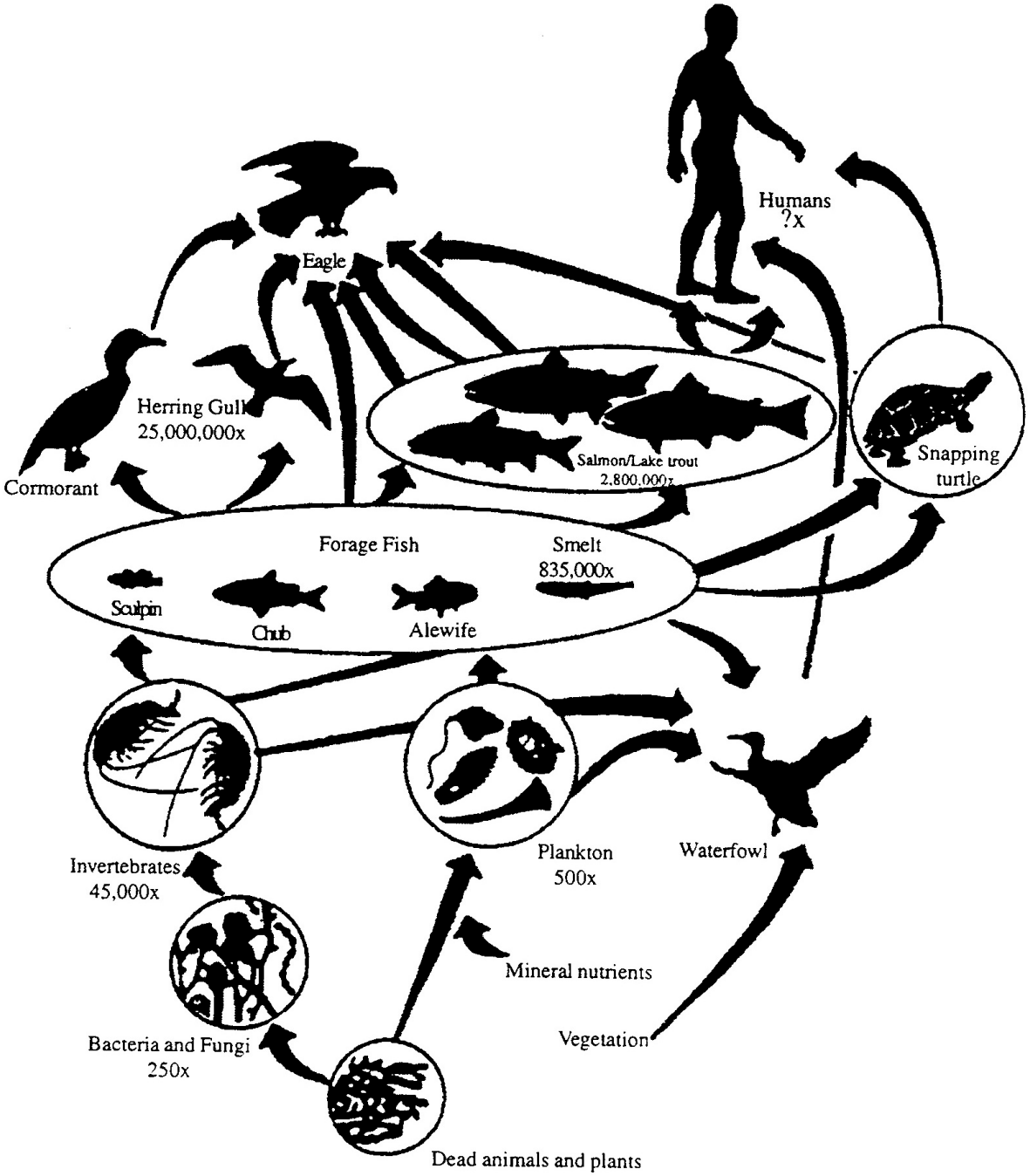
Organic Compounds	Metals
Benzo{a}pyrene	Alkylated Lead
DDT and metabolites	Mercury
Dieldrin	
Dioxins	
Furans	
Hexachlorobenzene	
Mirex	
PCBs	
Toxaphene	

Adapted from IJC 1998.

1.3 Pathways of Exposure and Human Health

The chemical pollutants of concern in Lake Michigan include substances such as organochlorines and heavy metals that are known to cause adverse health effects in animals and humans (Table 3). Organochlorines are highly lipophilic and biomagnify in the aquatic food chain, making them available to humans and other higher forms of life, as shown in Figure 1. Lipophilic substances found in the Great Lakes basin include PCBs, hexachlorobenzene, DDT and metabolites, dioxin (2,3,7,8-TCDD), mirex, dieldrin, toxaphene, and furans (Johnson et al. 1999). In addition, alkylated lead and mercury also tend to bioaccumulate in human tissue and are known to cause harmful health effects (Johnson et al. 1999). Recent findings indicate that populations continue to be exposed to persistent toxic substances within the Great Lakes and St.

Figure 1: Great Lakes Food Web



Great Lakes food web. Heavy metals and many synthetic chemicals are absorbed by organisms and bioaccumulate, with concentrations reaching toxic levels if exposure is great enough. The concentration is magnified at each step of the food web as large organisms eat many small ones. Numbers = Biomagnification of PCBs in Lake Ontario.
Source: Johnson et al. 1999.(reprinted with permission)

Lawrence River basins and that health consequences are associated with these exposures (Johnson et al. 1999).

Although the contaminant levels in the Great Lakes are declining, in general, recent trends suggest that concentrations for some pollutants may be leveling off. Low or declining levels may be associated with health consequences. For example, Jacobson and coworkers (1984) found that chronic, low-level prenatal exposure to PCBs was associated with neurobehavioral deficits in infants whose mothers consumed contaminated Lake Michigan fish. These results are supported by the recent work by Lonky and coworkers (1996) in newborns.

The three major routes that chemical and microbial pollutants enter the human body are by ingestion (water, food, soil), inhalation (airborne particles), and dermal contact. The human health goals for the Lake Michigan LaMP are driven by improvements in beneficial uses that are related to these exposure pathways. For example, restrictions on fish and wildlife consumption, restrictions on drinking water consumption, or beach closings prevent populations in the Lake Michigan area from enjoying the beneficial uses of the lake. An awareness of the underlying causes of these restrictions (e.g., chemical and microbial contaminants) and their associated health consequences will allow public health agencies to develop societal responses protective of public health. The human health goals for the Lake Michigan LaMP are outlined in Table 2.

Table 2: Human Health Goals for Lake Michigan LaMP and Pathways of Exposure

Human Health Goal	Pathway of Exposure
We can all eat any fish	Ingestion of food (fish)
We can all drink the water	Ingestion of water
We can all swim in the water	Dermal contact, inhalation, ingestion

1.4 Chemical Pollutants and Microbial Pollutants

The Lake Michigan LaMP has also established lists of Critical Pollutants, Pollutants of Concern, and Emerging Pollutants (Table 3). The Lake Michigan Critical Pollutants are chemicals that violate the most stringent Federal/State water quality standard or criteria, exceed

the U.S. Food and Drug Administration (FDA) action levels in Lake Michigan fish, or are associated with lakewide use impairments. They are chlordane, DDT and metabolites, dieldrin, dioxins, furans, PCBs (polychlorinated biphenyls), and mercury. The Lake Michigan Pollutants

Table 3: Lake Michigan Pollutants

Critical Pollutants	Pollutants of Concern	Emerging Pollutants
Chlordane	Arsenic	Atrazine
DDT & Metabolites	Cadmium	Selenium
Dieldrin	Chromium	PCB Substitute Compounds
Dioxins	Copper	DEHP (see text)
Furans	Cyanide	TCBT (see text)
PCBs	Hexachlorobenzene	
Mercury	Lead - alkylated	
	PAHs (see text)	
	Toxaphene	
	Zinc	

of Concern are defined as toxic substances associated with local or regional use impairments, or those for which there is evidence that loadings to or ambient concentrations in the Lake Michigan watershed are increasing. They are arsenic, cadmium, chromium, copper, cyanide, hexachlorobenzene, lead, PAHs (polycyclic aromatic hydrocarbons), toxaphene, and zinc. The Lake Michigan Emerging Pollutants are toxic substances that, while not presently known to contribute to impairments or to show increasing loadings/concentrations, have characteristics that indicate a potential to impact the physical or biological integrity of Lake Michigan. They are atrazine, PCB substitute compounds, such as DHEP (di(2-ethylhexyl)phthalate) and TCBT (tetrachlorobenzyltoluene), and selenium. In addition to examining the chemical pollutants, the Lake Michigan LaMP Human Health Appendix will also examine microbial pollutants in recreational waters and drinking water.

1.5 Weight of Evidence Approach

The weight of evidence approach will be used to describe the wildlife and laboratory studies supporting existing humans studies which demonstrate the potential human health impact from exposure to chemical pollutants (Johnson et al. 1998). The objectives of the Lake Michigan LaMP Human Health Appendix will be accomplished by utilizing peer-reviewed reports published by the Agency for Toxic Substances and Disease Registry (ATSDR) and other organizations.

2.0 Lake Michigan Pollutants

Toxicological summaries for Lake Michigan Critical Pollutants, Pollutants of Concern, and Emerging Pollutants are provided below.

2.1 Toxicology of Chemical Pollutants

2.11 Critical Pollutants

1. *Chlordane* (ATSDR 1995a)

   Chlordane is a manufactured chemical that was used as a pesticide in the United States from 1948 to 1988. In 1983, the U.S. Environmental Protection Agency (USEPA) banned all uses of chlordane except to control termites. In 1988, USEPA completely banned all uses. Chlordane adheres strongly to soil and is not likely to enter groundwater. Chlordane most commonly leaves soil by evaporation into the air. It builds up in the tissues of fish, birds, and mammals. Chlordane affects the nervous system, the digestive system, and the liver in animals and humans. Headaches, irritability, confusion, weakness, vision problems, vomiting, stomach cramps, diarrhea, and jaundice have occurred in persons who breathed air containing high concentrations of chlordane or accidentally swallowed small amounts of chlordane. Large amounts of chlordane taken by mouth can cause convulsions and death in humans. Mice fed low levels of chlordane in food developed liver cancer. Studies of workers who made or used chlordane do not show that exposure to chlordane is related to cancer, but the information is not sufficient to know for certain. The International Agency for Research on Cancer has determined

that chlordane is not classifiable as to its carcinogenicity to humans.

2. *DDT, DDE, and DDD* (ATSDR 1995c)

DDT [1,1,1-trichloro-2,2-bis(p-chlorophenyl)ethane] is a manufactured chemical widely used to control insects on agricultural crops and insects that carry diseases like malaria and typhus. It does not occur naturally in the environment. Because of adverse health effects to wildlife and the potential harm to human health, the use of DDT was banned in the United States. DDT is still used in other countries. Two similar metabolites of DDT are DDE [1,1-dichloro-2,2-bis(p-chlorophenyl)ethylene] and DDD [1,1-dichloro-2,2-bis(p-chlorophenyl)ethane]. DDD was also used to kill pests, but its use has also been banned. DDT adheres strongly to soil and does not move quickly to underground water. It is broken down quickly in air. DDT in soil usually breaks down to form DDE or DDD. DDT builds up in plants and in the fatty tissues of fish, birds, and animals. DDT affects the nervous system. Persons who accidentally swallowed large amounts of DDT have developed excitability, tremors, and seizures. These effects disappeared after the exposure ceased. Persons administered 6 mg DDT per kilogram of body weight orally by capsule generally demonstrated no neurological illness. Long-term occupational exposures to DDT have resulted in reversible changes in the levels of liver enzymes that influence the liver function. In animals, short-term exposure to large amounts of DDT in food affected the nervous system. In animals, long-term exposure to DDT has affected the liver. Animal studies suggest that short-term exposure to DDT in food may have a harmful effect on reproduction. The U.S. Department of Health and Human Services has determined that DDT may reasonably be anticipated to be a human carcinogen. The USEPA has determined that DDE and DDD are probable human carcinogens.

3. *Dieldrin* (ATSDR 1993a)

Aldrin and dieldrin are insecticides having similar chemical structures. Sunlight and bacteria convert aldrin to dieldrin, so that dieldrin is found more frequently in the environment than aldrin. In 1974, USEPA banned the use of aldrin and dieldrin, due to the potentially harmful effect to human health and the environment. Since the ban, very little dieldrin has been found in most foods; however, fish, seafood, dairy products, fatty

meats, and root crops from contaminated water or soil, may still have higher levels of dieldrin. The potential effects from low levels of exposure to aldrin and dieldrin are not known. However, headaches, dizziness, vomiting, irritability, and uncontrolled muscle movements have been observed in occupational pesticide applicators. Ingestion of moderate levels of these substances may accumulate in the body and cause convulsions. Exposure to very high levels due to accidental or intentional ingestion of aldrin and dieldrin may lead to convulsions and death. Although mice exposed to high levels of dieldrin are reported to develop liver cancers, no such evidence has been documented in humans. The International Agency for Research on Cancer has determined that aldrin and dieldrin are not classifiable as to their carcinogenicity to humans.

4. *Dioxins* (ATSDR 1999a)

Chlorinated dibenzo-p-dioxins (CDDs) are a family of 75 chemically related compounds, commonly known as dioxins. Of these, 2,3,7,8-TCDD (2,3,7,8-tetrachlorodibenzo-p-dioxin) is the most toxic and the most studied. Dioxins, particularly 2,3,7,8-TCDD, are formed in paper and pulp manufacture, during chlorination of waste and drinking water, and in the manufacture of organic chemicals. However, dioxins as a byproduct of chlorination have not been found in Michigan drinking water (Communication from Michigan Department of Community Health 3/24/00). Air releases occur from solid waste and industrial incineration. CDDs escape from waste water and are broken down by sunlight, evaporate into the air, but generally adhere to soil and become part of the water sediment. CDDs can be found at measurable concentrations in animals within the food chain due to bioaccumulation. More than 90% of the intake of CDDs in the general population comes from consumption of meat, dairy products, and fish. Chloracne (acne-like skin lesions) of the face and upper body occurs from exposure to large amounts of 2,3,7,8-TCDD, and other skin effects are also seen (i.e., skin rash, discoloration, and body hair). Blood and urine changes may be an indication of liver damage. Exposures to high concentrations of CDDs may result in long-term changes in glucose metabolism and subtle changes in hormonal levels. Animal studies have also demonstrated an increased risk of cancer from 2,3,7,8-TCDD. Studies in humans suggest that 2,3,7,8-TCDD

exposure increases the risk of several types of cancer. The U.S. Department of Health and Human Services has determined that 2,3,7,8-TCDD is known to cause cancer.

5. *Chlorodibenzofurans* (CDFs) (ATSDR 1995b)

CDFs are a family of chemicals (135) containing 8 chlorine atoms attached to the parent chemical dibenzofuran. The CDFs with chlorine atoms at the 2,3,7,8-positions are particularly harmful. CDFs are by-products of paper and pulp manufacture and incineration. They are generally found in air as solid particles and vapors. Snow and rain can remove the vaporized CDFs from air. They attach to soil, sediments in lakes and rivers, and may bioaccumulate in fish at levels much greater than detected in water or sediment. CDFs can also bioaccumulate in animals, birds, and humans through ingestion of contaminated foods. Ninety-percent (90%) of the daily exposure comes from consumption of contaminated food (e.g., meat, fish, milk). Inhaling air or drinking water, and contact with contaminated soil are other sources of exposure. Most of the data on human health comes from studies of accidental exposure to foods contaminated with CDFs. The concentration of CDFs, in these cases, exceeded the amounts from environmental or dietary exposure. CDFs exposures resulted in skin and eye irritation (e.g., acne, skin discoloration, swollen eyelids), vomiting and diarrhea, anemia, lung infections, numbness, nervous system effects, and mild liver changes. It is unknown if CDFs cause human cancer. The U.S. Department of Health and Human Services has not classified CDFs for carcinogenicity.

6. *PCBs* (polychlorinated biphenyls) (ATSDR 1997c)

PCBs are organic chemicals consisting of 209 individual congeners. Some commercial mixtures in the United States have the industrial trade name of Aroclor. PCBs have been used as coolants and lubricants in transformers, capacitors, and other electrical equipment. PCBs manufacture ceased in 1977 due to accumulation of PCBs in the environment and potential adverse health effects. Today, PCBs can be discharged into the environment from hazardous waste sites containing PCBs, illegal dumping, and leaks from transformers. PCBs can travel in air. In water, most PCBs adhere to organic particles and sediments with a small amount dissolved in the water. Fish and marine

mammals may bioaccumulate PCBs in excess of the amounts found in water. Human exposure may result from eating food (e.g., fish, meat, dairy products), drinking well water contaminated with PCBs, breathing air near hazardous waste sites, or leaks of PCBs from appliances. PCBs have been associated with birth defects and reproductive problems in human studies. Inhalation of PCBs results in irritation of the nose and lungs, and skin irritations. PCBs cause liver cancer in animals, but it is not known if PCBs are a human carcinogen. The U.S. Department of Health and Human Services has determined that PCBs may reasonably be anticipated to be carcinogens.

7. *Mercury* (ATSDR 1999b)

Mercury is a metal found naturally in the environment. It can be found in the metallic, liquid form; as inorganic mercury compounds (salts) with chlorine, sulfur, or oxygen; and as organic mercury in combination with carbon. Methylmercury is the most common organic mercury. Metallic mercury is utilized in the production of chlorine gas and caustic soda, and is used in thermometers, dental fillings, and batteries. Mercury salts have been used in skin lightening creams and as antiseptic creams and ointments. Inorganic mercury is found in air from mining, burning of coal and waste, and from industry, and can also be found in water or soil as natural deposits, waste disposal, and volcanic activity. Methylmercury is produced by the action of microscopic organisms in water and soil. It can bioaccumulate in fish with the larger and older fish having the highest levels of mercury. Exposure to methylmercury may occur from consuming contaminated fish or shellfish; inhalation of vapors from spills, incinerators, and industry; or inhaling contaminated air or skin contact in the workplace. The nervous system is susceptible to all forms of mercury. Because methylmercury and metallic mercury vapors can affect the brain, they are more harmful than other forms of mercury. Exposure to high levels of metallic, inorganic, or organic mercury can permanently injure the brain, kidneys, and developing fetus. Mercury in mother's breast milk may be passed to the nursing infant. Nervous system effects include irritability, shyness, tremors, changes in vision or hearing, and memory problems. Short-term exposure to metallic mercury vapors at high concentrations may produce lung damage, nausea and vomiting, diarrhea,

rise in blood pressure or heart rate, skin rashes, and eye irritation. Effects of fetal exposure include incoordination, blindness, seizures, and inability to speak. Children exposed to mercury may exhibit effects on the nervous and digestive systems and to the kidney. Animal studies have shown tumor development from exposure to mercuric chloride and methylmercury. The data on cancer effects is inadequate for human exposure to mercury. USEPA has concluded that mercury chloride and methylmercury are possible human carcinogens. The U.S. Department of Health and Human Services and the International Agency for Research on Cancer have not classified mercury as to its human carcinogenicity.

2.12 Pollutants of Concern

1. *Arsenic* (ATSDR 1993b)

Arsenic is present in the environment in two forms, organic and inorganic. Organic arsenic is formed by combining with carbon and hydrogen and is less harmful than inorganic arsenic compounds, which are formed in combination with oxygen, chlorine, and sulfur. Inorganic arsenic is used most often in the production of wood preservatives, insecticides, and weed killers. Although arsenic bioaccumulates in fish and shellfish, it is generally in the less harmful organic form. Human exposure may occur by breathing sawdust or burning smoke from arsenic-containing wood; by breathing workplace air where arsenic is detected; and by ingesting contaminated water, soil, or air at waste sites or places with high detected levels of arsenic. Inorganic arsenic is a human poison. Ingesting high levels of inorganic arsenic in food or water can be fatal. Other health effects from high-level exposure include damage to tissues, and development of a sore throat and irritated lungs. Low-level exposure may lead to nausea, vomiting, diarrhea, decreased production of red and white blood cells, abnormal heart rhythm, blood vessel damage, and “pins and needles” sensations in the hands and feet. Inhaling inorganic arsenic increases the risk of lung cancer. Ingestion increases the risk of skin cancer and tumors of the bladder, kidney, liver, and lung. The U.S. Department of Health and Human Services has determined arsenic to be a known carcinogen.

2. *Cadmium* (ATSDR 1993c)

Cadmium may be found naturally in the earth's crust, usually combined with other elements. It can be found in water and soil from waste disposal, spills, or leaks from hazardous waste sites. Fish, plants, and animals take in cadmium from the environment. Human exposure may result from inhalation of cadmium in the workplace or from municipal waste sites, or cigarette smoke. It can also result from ingestion of contaminated water, fish, plants, and animals. Low level, long term exposures from air, food, or water may result in kidney disease. High level inhalation exposure can result in severe lung damage and death. Ingestion of very high levels in food or drinking water may result in vomiting and diarrhea. Based upon weak human and strong animal evidence, the U.S. Department of Health and Human Services has determined that cadmium and its compounds may be carcinogenic.

3. *Chromium* (ATSDR 1993d)

Chromium is a naturally occurring element found in most mineral and organic substances. It is used in the production of steel and other alloys, bricks, dyes and pigments, and for chrome plating, leather tanning, and wood preserving. Chromium enters the environment when these products, or fossil fuels, are burned. Dissolved chromium is not common, as the element tends to remain bound to particles in the water column. Fish and other aquatic life do not bioaccumulate chromium. Chromium can be toxic at high levels. Human exposure occurs mainly through breathing air or sawdust from industries which manufacture chromium-containing products, or at waste sites where chromium exists at high levels. Ingesting food or water contaminated with chromium from soil (near such sites) could also lead to exposure. High levels of chromium ingestion can be fatal, or could lead to stomach upsets, ulcers, convulsions, kidney and liver damage. Breathing high levels of chromium can cause damage or irritation to the nose, lungs, stomach, and intestines. Long-term exposure to high levels of chromium leads to nose and lung damage. In addition, the risk of non-cancer lung diseases increases with exposure level. The U.S. Department of Health and Human Services has determined that certain chromium compounds are carcinogens.

4. *Copper* (ATSDR 1990)

Copper may be found naturally in the earth's crust, water, air as well as in plants and animals. This common environmental element is essential for all living organisms. It can be found in mixtures of metals, natural occurring minerals and man-made chemicals. Mined and processed extensively in the U.S., copper is used in the manufacture of metals, industry, agriculture, and as a preservative for wood, leather and fabrics. Exposure may result from ingestion, inhalation and dermal contact. Individuals living or working near copper mines or processing facilities may breathe higher levels of copper containing dust. Individuals in households with copper pipes and brass faucets may be exposed to high levels of copper in drinking water. However, copper found in lakes and rivers does not easily enter the body since it strongly adheres to existing particles in lake and river water. Copper may be deposited in soil from copper production facilities, mining, and sewage treatment plant sludge. It adheres strongly to soil. Although copper is essential for good health, high levels of copper may be harmful. Long-term exposure may result in irritation of the nose, mouth, and eyes and can cause headaches, dizziness, nausea, and diarrhea. Drinking water with high levels (greater than 1.3 ppm) of copper may cause vomiting, diarrhea, stomach cramps, and nausea. Liver and kidney damage and death may result if high levels (greater than 2-3 milligrams for adults) of copper are intentionally consumed. Copper has not been classified as a carcinogen.

5. *Cyanide* (ATSDR 1997a)

Cyanide is a chemical produced naturally and as a by-product of electroplating, metallurgy, chemical production, photographic development, plastic production, ship fumigation, and some mining processes. Cyanide and the chemicals containing cyanide enter the environment through natural and industrial processes. Airborne cyanide exists as a gas, or adhered to fine dust particles. Cyanide in surface water typically evaporates as hydrogen cyanide. High concentrations of cyanide in the soil, however, can pass through to groundwater. Once in the water column, cyanide will not accumulate in fish or other aquatic organisms. Human exposure to cyanide occurs by breathing contaminated air such as cigarette smoke, smoke from fires, air near a hazardous waste site, or air inside an

industrial facility which produces cyanide as a by-product. Cyanide may also be ingested by eating contaminated foods, or touching contaminated soil or other surfaces. Short-term exposure to high levels of cyanide in the air can adversely affect the heart and brain, and can cause coma and death. Low level, long-term cyanide exposure can lead to difficulty breathing, heart pain, vomiting, blood changes, headaches, and thyroid gland enlargement. According to the USEPA, cyanide has not been classified as a human carcinogen.

6. *Hexachlorobenzene* (ATSDR 1997b)

Hexachlorobenzene is a compound which does not occur naturally in the environment. Rather, it is formed as a by-product of chemical production processes, and may be found in the waste streams of chloralkali and wood-preserving plants, and when burning municipal waste. Hexachlorobenzene is found in the environment, most often, bound to soil particles, as it does not dissolve easily in water. It does accumulate in plants such as wheat and grasses as well as organisms including fish, birds, lichens and mammals which feed on lichens. Exposure to hexachlorobenzene can be harmful to humans. Human exposure generally occurs by ingesting contaminated food such as fish, milk, dairy products, meat from cattle which grazed on contaminated pastures, breast milk from exposed mothers, or water, or through breathing contaminated air or touching soil or other contaminated substances. Ingestion of hexachlorobenzene may lead to damage of the liver, thyroid, nervous system, bones, kidneys, blood, and immune and endocrine systems. The U.S. Department of Health and Human Services has determined that hexachlorobenzene may reasonably be expected to be a carcinogen.

7. *Lead* (ATSDR 1993f)

Lead is a metal found naturally occurring in the earth's crust. It enters the environment through mining, the burning of fossil fuels, and manufacturing batteries, ammunition, metal products, roofing, and devices to shield x-rays. Lead binds to soil particles and remains in the air after emission until the particles fall to the ground or water. Lead in urban soil can also be attributed to landfills and leaded paint. Acidic water can cause lead to dissolve into the water column from contaminated sediments. Almost every organ and

system in the human body may be affected by lead. Humans are exposed to lead by breathing contaminated air from industry, tobacco smoke, or hobbies, and, also, ingesting lead-based paint chips, food grown on contaminated soil or food covered with contaminated dust, and drinking water from lead pipes or pipes with lead soldering. The central nervous system is particularly sensitive, and the kidneys and immune system are at risk as well. Children and fetuses are particularly sensitive to high level exposure, with dangers of premature births, low birth weights, decreased mental abilities, learning difficulties, and reduced growth. Adults may suffer decreased reaction times, weakness, anemia, and reproductive disorders when exposed to high levels of lead. The U.S. Department of Health and Human Services has determined that lead acetate and lead phosphate may reasonably be anticipated to be carcinogenic.

8. *Polycyclic Aromatic Hydrocarbons* (PAHs) (ATSDR 1996)

PAHs consist of over 100 chemicals which are formed during the incomplete combustion of fossil fuels, municipal waste, and other organic substances. Some PAHs are manufactured for use in coal and roofing tar, crude oil, dyes, plastics, and pesticides. PAHs enter the environment through the combustion of fossil fuels as well as from volcanoes and forest fires. Generally, PAHs bind to soil particles, so are found in airborne particles as well as in sediment in the water column. Plants and animals do accumulate PAHs, and the concentration in these organisms may be much greater than the concentration in surrounding water or soil. PAHs are considered harmful to human health. Humans may be exposed to PAHs by breathing contaminated air from industrial processes or contaminated smoke; having physical contact with air, water, or soil near hazardous waste sites; eating contaminated foods or drinking contaminated water or milk; and breast milk for infants of mothers exposed to PAHs. No harmful effects have been proven in humans, although animal studies have shown adverse effects on the reproductive cycle, skin, body fluids, and the ability to fight disease. The U.S. Department of Health and Human Services has determined that some PAHs may reasonably be expected to be carcinogens.

9. *Toxaphene* (ATSDR 1997e)

Toxaphene is used as an insecticide and is a mixture of over 670 chemicals. Its usual state is as a solid or gas. Prior to its cancellation (1982) and ban (1990), toxaphene had a high-volume use as an insecticide. One of its uses was the destruction of unwanted fish in lakes. Toxaphene may be found in the environment from migration near hazardous waste sites and may evaporate into the air. Since it does not dissolve easily in water, it is generally found in soil or sediment at the bottom of lakes or streams, or in the air. Toxaphene deteriorates slowly in the environment and bioaccumulates in fish and mammals. Exposure may also result from inhalation of contaminated air near hazardous waste sites, or may be ingested by children eating contaminated soil. Exposure may also result from consuming fish and shellfish, or well water contaminated with toxaphene. Inhalation or consumption (eating or drinking) high levels of toxaphene may result in injury to the lungs, nervous system, and kidneys, and may be fatal. Human health studies are not available. However, animal studies have shown developmental effects in newborns, whose mothers received exposure to toxaphene during pregnancy. The U.S. Department of Health and Human Services has determined that toxaphene may reasonably be anticipated to be a human carcinogen.

10. *Zinc* (ATSDR 1995d)

Zinc is an element found in the earth's crust. Most zinc is released into the environment through mining, steel production, coal burning, and burning of waste. Zinc exists in the air bound to particles, but dissolves easily in water. Zinc does accumulate in fish and other organisms but not in plants. Although zinc is an essential component in the human diet, it can be harmful to human health in large quantities. Humans are exposed to zinc through drinking water contaminated at manufacturing or waste sites; water or other liquids stored in, or transported through, zinc-coated pipes or containers; breathing zinc particles at manufacturing sites; or eating too many zinc dietary supplements. High levels of zinc consumption can cause stomach cramps, nausea, and vomiting. Long-term zinc over-consumption can lead to anemia, pancreas damage, and low levels of high density lipoprotein cholesterol. Breathing high levels of zinc can cause a short-term disease called

metal fume fever which affects the lungs and body temperature. The U.S. Department of Health and Human Services, the International Agency for Research on Cancer, and the USEPA have not classified zinc for carcinogenicity.

2.13 Emerging Pollutants

1. *Atrazine* (EPA 1999a; Sittig 1991)

   Atrazine is one of a group of chemically similar triazine herbicides. They include cyanazine, propazine, and simazine, as well as atrazine. At present, atrazine is one of the two most utilized U.S. agricultural pesticides. Exposure may result from ingesting food and drinking water contaminated with the triazines, or occupationally in the application of pesticides. Harmful health effects include dermatitis and eye irritation. High exposure levels may affect the nervous system. USEPA has classified atrazine and the other triazines as possible human carcinogens.

2. *PCB Substitutes:*

   DEHP (ATSDR 1993e)

   DEHP [di(2-ethylhexyl)phthalate] is a manufactured chemical used in the production of polyvinyl chloride plastic products. It is also found in inks, pesticides, cosmetics, and vacuum pump oil. Because of its use in plastics, it is ubiquitous in the environment. It can dissolve in water more quickly in the presence of gas, oil, or paint removers, and adheres well to soil particles. Microorganisms in water or soil help to break down DEHP into harmless compounds. This break down does not occur easily in soil, or in lake or river bottoms. DEHP can be found in plants, fish, and other animals; however, animals high on the food chain have the ability to break down DEHP, avoiding accumulation in the body tissues. Exposure, usually at low levels, may occur from plastic medical products and food wrap, in well water near waste sites, occupational exposure, or tubing for kidney dialysis. Evidence does not show that DEHP causes serious human health effects. In animals, airborne exposure did not produce serious health effects. Very high exposure of animals in food or water caused sperm damage which was reversed when the exposure ceased. Longer exposure caused reproductive and birth defects in male and

female animals. Long-term, high level exposures caused kidney damage to animals, similar to that seen in long-term dialysis patients. Dermal effects are unlikely because it is not absorbed through the skin. The U.S. Department of Health and Human Services has determined that DEHP may reasonably be anticipated to be a human carcinogen.

TCBT

TCBT (tetrachlorobenzyltoluene) is a PCB substitute that induces toxicological changes similar to PCBs with pathological effects to the liver and thyroid in mice (Murk et al. 1991). Hepatic toxicity was also observed in rats with introduction of TCBT by gavage (Bouraly and Millischer 1989). Rats exposed to a single high oral dose (300 mg/kg) had a marked rise in the liver's cytochrome-P-450c dependent reactions (von Meyerinck et al. 1990).

3. *Selenium* (ATSDR 1997d)

Metallic selenium is usually found in rocks and soil. Selenium in rock is usually combined with sulfide minerals or with silver, copper, lead, and nickel minerals. It is also found combined with oxygen. Selenium sulfide is found in anti-dandruff shampoos. Selenium acid, formed from selenium dioxide, is used to clean guns. Selenium can be found in air or in irrigation drainage water from agricultural fields. It can accumulate in animals residing in areas where water is contaminated with selenium. Exposure may result from inhaling contaminated air or by consuming food, water, or dietary supplements containing selenium. Exposure to high levels has produced dizziness, fatigue, irritation, fluid in the lungs, and severe bronchitis. The exact levels causing these effects is not known. Skin contact may result in rashes, swelling, and pain. Dietary levels higher than 5-10 times the daily requirement can be harmful. Excessive doses can be life threatening, result in brittle hair and deformed nails, and also loss of feeling and control in the arms and legs. Animal studies have reported liver and lung tumors in animals at high doses. The U.S. Department of Health and Human Services has determined that selenium sulfide is reasonably anticipated to be a carcinogen.

2.2 Recreational Waters

In 1972, the US Congress passed the Clean Water Act to improve the polluted condition of the nation's waters (EPA 1998). Although the Act's original intent was to have "fishable and swimmable" water, presently, its main objective is to preserve the "chemical, physical and biological integrity of water". The most significant source of water pollution is polluted runoff (e.g., oxygen depleting substances, metals, pesticides, organic chemicals). Control of pollution from point sources, such as factories and city sewers has helped to reduce contamination. In addition, the Act has been responsible for the twofold increase in waterways that are safe for swimming and fishing. However, assessments by states indicate that 40 % of the nation's waterways are still not safe for fishing and swimming.

Beaches, rivers, and lakes are the destinations most utilized by Americans as vacation sites (NRDC 1999) . During these trips, Americans will swim, fish, go boating, or relax. Swimming involves full body contact with the water, and may involve ingestion or inhalation exposure to pollutants in water.

The Great Lakes Water Quality Agreement (IJC 1994) calls for recreational waters to be substantially free from bacteria, fungi, and viruses. These microbial organisms of fecal origin have the potential for causing relatively mild illnesses (e.g., gastroenteritis) to more serious illnesses (e.g., hepatitis, typhoid fever) from a single episode of exposure. The risk of illness is dependent upon the degree of water pollution, the individual's level of exposure, immunization status (e.g., polio), and the general health of the individual. For this reason, the protection of the public's health is directed at controlling microbial pollutants in recreational waters. See Table 4 for the swimming associated illnesses.

As a result of the Clean Water Act, the USEPA has established guidelines on the maximum level of pollution acceptable for fresh water use. The USEPA recommends enterococcus or E. coli as indicators because the detection of these organisms provides a reliable estimate of the presence of disease-causing bacteria or viruses in the water (NRDC 1999). Most states utilize the fecal coliform or total coliform counts to monitor recreational waters. For Great

Table 4: Pathogens and Swimming-Associated Illnesses

Pathogenic Agent	Disease
Bacteria	
E. coli	Gastroenteritis
Salmonella typhi	Typhoid fever
Other salmonella species	Various enteric fevers (often called paratyphoid), gastroenteritis, septicemia (generalized infections in which organisms multiply in the bloodstream)
Shigella dysenteriae and other species	Bacterial dysentery
Vibrio cholera	Cholera
Viruses	
Rotavirus	Gastroenteritis
Norwalkvirus	Gastroenteritis
Poliovirus	Poliomyelitis
Coxsackievirus (some strains)	Various, including severe respiratory diseases, fevers, rashes, paralysis, aseptic meningitis, myocarditis
Echovirus	Various, similar to coxsackievirus (evidence is not definite except in experimental animals)
Adenovirus	Respiratory and gastrointestinal infections
Hepatitis	Infectious hepatitis (liver malfunction), also may affect kidneys and spleen
Protozoa	
Cryptosporidium	Gastroenteritis
Giardia lambia	Diarrhea (intestinal parasite)
Entamoeba histolytica	Amoebic dysentery, infections of other organisms
Isopora belli and Isopora hominus	Intestinal parasites, gastrointestinal infection
Balantidium coli	Dysentery, intestinal ulcers

Source: NRDC 1999.

Lakes swimming (fresh waters), the EPA-recommended standard is a geometric mean of 33 enterococcus bacteria per 100 ml (milliliters), or 126 E. coli bacteria per 100 ml of water (EPA 2000). Of the eight Great Lakes states, Indiana, Michigan, and Ohio utilize the E. coli indicator to determine bacterial quality in freshwater. Illinois, Minnesota, New York, Pennsylvania, and Wisconsin use the fecal coliform as an indicator. New York also employs the total coliform count, as well as fecal coliform, as an indicator of fecal contamination.

In 1998, sewage spills and overflows, polluted runoff and stormwater, and rain were the major causes of pollution causing U.S. beach closings and health advisories (NRDC 1999). The eight Great Lakes states have monitoring and notification programs for alerting the public about polluted recreational waters. Illinois, Indiana, Ohio, and Pennsylvania have established comprehensive monitoring programs for most (or all) of their beaches and the means to notify the public of the results. Michigan, Minnesota, Wisconsin, and New York have regular monitoring and notification of the public for some of their recreational beaches. Indiana, Illinois, Ohio, and Pennsylvania close beaches when bacterial water-quality standards have been exceeded.

Because of the serious nature of some of the microbial diseases and the fact that 40% of the U.S. waterways are not safe for fishing and swimming, comprehensive monitoring and notification programs are needed to protect the public health.

2.3 Drinking Water

In 1974, Congress passed the Safe Drinking Water Act to regulate the US public drinking water supply (EPA 1999b). The Amendments of 1986 and 1996 mandated actions to protect public drinking water and its sources (i.e., rivers, lakes, reservoirs, springs, and ground water wells). The Great Lakes Water Quality Agreement (IJC 1994) also calls for the control of contaminated ground water.

The USEPA has been authorized by the Act to establish national health-based standards for drinking water to protect the public from man-made and naturally-occurring contaminants in drinking water (EPA 1999b). These standards are based on a health goal that has a risk basis for the general public and sensitive groups. Sensitive groups such as infants, children, pregnant women, the elderly, and immuno-compromised persons are considered when setting these health

goals. When the standards (i.e., legal limit of a contaminant in drinking water) are being established, they are set as close to the health goal as possible. The current standards for inorganic and organic chemicals, radionuclides, and microorganisms, and for Great Lakes pollutants can be found at the USEPA web site (http://www.epa.gov/safewater/mcl.html). The Act also requires water suppliers to notify the public about serious problems with water quality. USEPA also prepares an annual report on water system compliance. States are also required to produce and disseminate reports on water systems.

Although the USEPA works with states and water systems to ensure that these standards are being met, substances from various sources may pollute drinking water making it unfit to consume. These sources include improperly disposed chemicals, animal and human waste, pesticides, waste permeating underground, and natural substances (EPA 1999b). Other sources of drinking water contamination may include improper treatment or disinfection, or a faulty distribution system.

One example of improper treatment or disinfection of water occurred in Milwaukee Wisconsin in the spring of 1993. A water treatment plant failure resulted in the contamination of the city's water supply with Cryptosporidium parvum, a parasitic organism of fecal origin. An estimated 403,000 Milwaukee residents developed gastrointestinal illness due to the water contamination (Oswe et al.1996). The immuno-compromised seemed particularly vulnerable with high death rates (Hoxie et al. 1997). During the outbreak, the media succeeded in disseminating health information to the public (Cordell et al. 1997).

At present, the USEPA is assessing health risks from microbial contaminants (e.g., Cryptosporidium), byproducts of drinking water disinfection (i.e., trihalomethanes), radon, arsenic, and vulnerable ground water sources (EPA 1999b). These measures, the regulatory standards, and the notification of the public regarding serious problems are measures to ensure the safety of the nation's public drinking water.

3.0 Weight of Evidence Approach

The purpose of this section is to review the wildlife, laboratory, and human studies that demonstrate the public health risk from exposure to persistent toxic substances found in the

Great Lakes basin. Because of the ethical issue of exposing humans to toxic substances and factors such as a small sample size and presence of multiple chemicals, human studies are often limited in their ability to establish a causal relationship between exposure to chemicals and potential adverse human health effect. The weight of evidence approach utilizes the available information from wildlife and controlled animal experiments to support the results of human studies. The use of wildlife data supposes that animals can act as sentinels for adverse effects observed in humans (Johnson and Jones 1992).

3.1 Wildlife Populations

Reproductive impairments have been described in avian, fish, and mammalian populations in the Great Lakes. For example, egg loss due to egg shell thinning has been observed in predatory birds, such as the bald eagle, within the Great Lakes (Menzer and Nelson 1980). After feeding on Great Lakes fish for two or more years, immigrant birds (eagles) were shown to have a decline in reproductive success (Colburn et al. 1993). Developmental effects in the form of congenital deformities (e.g., crossed mandibles, club feet) have also been reported in the avian population within the Great Lakes basin (Stone 1992).

Effects to the endocrine system and tumor formations have been detected in fish populations. Researchers have reported enlarged thyroids in all of the 2 to 4 year-old Great Lakes salmon stocks that were examined (Leatherland 1992). Tumors associated with exposure to high levels of polyaromatic hydrocarbon compounds have also been detected in brown bullhead fish in the Great Lakes area (Baumann et al. 1982).

Effects on the immune system have also been a notable finding. At a number of Great Lakes sites, a survey of herring gulls and Caspian terns demonstrated a suppression of T-cell-mediated immunity following prenatal exposure to organochlorine pollutants particularly PCBs (Grasman et al. 1996). For more examples of these effects in wildlife, see Table 5.

Table 5: Effects of Toxic Contamination on Fish and Wildlife in the Great Lakes

Species	Population decrease	Effects on Reproduction	Eggshell thinning	Birth defects	Behavioral changes	Biochemical changes	Mortality
Mink	✓	✓	NA	NE	NE	NE	✓
Otter	✓		NA	NE	NE	NE	S
Double-crested cormorant	✓	✓	✓	✓		✓	S
Black-crowned night heron	✓	✓	✓	✓		✓	S
Bald eagle	✓	✓	✓	NE		NE	NE
Herring gull		✓	✓	✓	✓	✓	✓
Ring-billed gull				✓		NE	✓
Caspian tern		✓		✓	NE	NE	
Common tern		✓	✓	✓		✓	
Forster's tern		✓		✓	✓	✓	
Snapping turtle	NE	✓	NA	✓	NE	NE	NE
Lake trout		✓	NA			✓	
Brown bullhead			NA			✓	
White sucker			NA	✓		✓	

Source: USEPA's National Water Quality Inventory: 1992 Report to Congress.

Notes: NA = not applicable

NE = not examined

S = suspected

3.2 Animal Experiments

Toxicology evidence in animals is mounting that supports the association between exposure to persistent toxic substances and adverse health effects. Animal experiments have demonstrated a wide range of health outcomes from exposure to PCBs, mercury and chlorinated dibenzo-p-dioxins (CDD).

PCBs (polychlorinated biphenyls): Animals exposed orally to PCBs developed effects to the hepatic, immunological, neurological, developmental and reproductive systems. Effects have also been reported in the gastrointestinal and hematological systems (ATSDR 1998). Animal ingestion studies strongly support the finding that higher chlorinated PCB mixtures (i.e., 60% chlorine by weight) are carcinogenic to the livers of rats, while the lower chlorinated PCBs are weaker animal carcinogens (i.e., lower incidence of total tumors and more benign tumors) (Buchmann et al. 1991, Sargent et al. 1992). A General Electric Company sponsored study demonstrated the carcinogenicity of Aroclor-1016, Aroclor-1242, Aroclor-1254, and Aroclor-1260 in rats receiving dietary exposure to PCBs. As an example, liver tumors were observed in female rats, and thyroid cancers were reported in male rats (Brunner et al. 1996).

A number of animal studies have demonstrated immune effects following exposure to PCBs (Arnold et al. 1995, Tryphonas 1995, Ross et al. 1996). In a laboratory study, habour seals were administered a diet of Baltic sea herring contaminated with organochlorine compounds and other pollutants (Ross et al. 1996). When compared with seals given a diet of relatively uncontaminated Atlantic Ocean fish, the seals ingesting the contaminated sea herring were found to have impaired natural killer cell activity and T-lymphocyte function.

Neurobehavioral effects have been seen in monkeys, exposed orally from birth to 20 weeks, to a PCB congener mixture representative of the PCB mixture found in the breast milk of Canadian women (Rice 1997). The monkeys were subsequently tested at 2.5 and 5 years of age, and found to have deficits in learning and difficulty in learning complex tasks when compared to controls.

Mercury: Long-term, high level animal ingestion exposure to mercury has been associated with cardiovascular (Arito and Takahashi 1991), developmental (Fuyuta et al. 1978, 1979; Nolen et al 1972; Inouye et al 1985), gastrointestinal (Mitsumori et al. 1990), immune

(Ilback 1991), renal (Yasutake et al. 1991; Magos et al. 1985; Magos and Butler 1972; Fowler 1972) and reproductive effects (Burbacher et al. 1988; Mitsumori et al.1990; Mohamed et al. 1987). The studies also indicate that the nervous system is particularly sensitive to mercury exposure by ingestion (Fuyuta et al. 1978; Inouye and Murakami 1975; Magos et al.1980, 1985). In addition, growth of kidney tumors has been reported in animals administered methylmercury in drinking water or diet for extended periods (Mitsumori et al. 1981, 1990).

CDDs (chlorinated dibenzo-p-dioxins): In specific species (e.g., guinea pig), very low levels of 2,3,7,8-TCDD (2,3,7,8-tetrachlorodibenzo-p-dioxin) have resulted in the death of the exposed animal after a single ingestion dose (NTP 1982). At nonlethal levels of 2,3,7,8-TCDD by ingestion, other effects reported in animals include weight loss (NTP 1982), biochemical and degenerative changes in the liver (NTP 1982, Kociba et al. 1978), and a decline in blood cells (Kociba et al 1978). Dermal effects in animals (e.g., hair loss, chloracne) have also been reported by ingestion exposure (Mc Connell et al. 1978). In many species, the immune system and fetal development are particularly susceptible to 2,3,7,8-TCDD exposure. Offspring of animals receiving oral exposure to 2,3,7,8-TCDD developed birth defects such as skeletal deformities and kidney defects, weakened immune responses, impaired reproductive system development, and learning and behavior impairments (Giavini et al. 1983, Gray and Ostby 1995, Tryphonas 1995, Schantz and Bowman 1989, Schantz et al. 1992). Reproductive effects in the form of miscarriages were reported in rats, rabbits, and monkeys exposed orally to 2,3,7,8-TCDD during pregnancy (McNulty 1984). Rats of both sexes were observed to have endocrine changes in the form of alterations in sex hormone levels with dietary exposure. Other reproductive effects include a decline in sperm production in male rats, and carcinogenic effects of cancer of the liver, thyroid, and other sites in rats and mice exposed orally to 2,3,7,8-TCDD (NTP 1982, Kociba et al. 1978). Research evidence is also increasing, supporting the neurotoxic effect for mammals and birds from ingestion exposure to dioxin-like compounds, including certain PCBs and CDFs. Changes in thyroid hormones and neurotransmitters, singly or together, at critical periods in the development of the fetus are considered responsible for the neurological changes (Brouwer et al. 1995; De Vito et al. 1995; Henshel et al. 1995b; Henshel and Martin 1995a; Vo et al. 1993).

3.3 Human Health Studies

3.3.1 Exposure Studies: Fish species residing in waters contaminated with lipophilic pollutants (i.e., fat-soluble pollutants as PCBs) bioaccumulate these contaminants and become a further source of contamination for larger, predator fish (e.g., sport caught trout and salmon) (Humphrey 1988). This process results in a biomagnification or increase in the levels of contaminants in the predator fish which may subsequently be consumed by humans. Fish consumption has been shown to be a major pathway of human exposure to persistent toxic substances such as PCBs (Birmingham et al. 1989; Fitzgerald et al. 1996; Humphrey 1983; Newhook 1988), exceeding exposures from land, air, or water sources (Humphrey 1988). Humphrey (1988) reported that PCBs were the dominant contaminants detected in Lake Michigan trout (3,012 parts per billion or ppb) and chinook and coho salmon (2,285 ppb) surpassing other contaminants such as DDT (1,505 ppb, 1,208 ppb), hexachlorobenzene (5 ppb, 5 ppb), oxychlordane (25 ppb, none shown), trans-nonachlor (195 ppb, 162 ppb) and dieldrin (75 ppb, 53 ppb) respectively in trout and salmon. Fish specimens collected from the dinner plate of study participants were used to determine these median PCB concentrations. Recently, total PCB levels have decreased in most Lake Michigan fish species and appear to remain below the FDA action level of 2 mg/kg (parts per million or ppm) but the concentrations in chinook and coho salmon have risen slightly since the late 1980s (Stow et al. 1995).

Early investigations of Lake Michigan fish consumption have broadened our knowledge about transmission of contaminants from fish to humans, including fetus and infant exposure from maternal consumption. Investigating a cohort of Lake Michigan fisheaters, Humphrey (1988) discovered that sport anglers who regularly consumed Great Lakes salmon and trout (consumption rate of $\geq$24 pounds/year [or $\geq$11 kg/year]) had median serum PCB levels approximately 4 times higher (56 ppb) than those who consumed no or very small amounts of Lake Michigan fish (15 ppb) (consumption rate of 0-6 pounds/year [or 0-2.7 kg/year]). Halogenated contaminants (e.g., PCBs) have also been detected in adipose tissue, breast milk, and cord blood, associated with consumption of contaminated fish (ATSDR 1998). Other studies

have also supported these findings. For example, Schwartz et al. (1983) demonstrated that consumption of Lake Michigan fish was positively associated with the PCB concentration in maternal serum and breast milk. Maternal serum PCB concentrations were also positively associated with the PCB levels in the umbilical cord serum of the infant (Jacobson et al. 1983).

Body burden levels of PCBs are still elevated in fish consumers. Hovinga et al (1992) reported a mean serum PCB concentration of 20.5 ppb in 1982 for persons consuming >24 pounds of Lake Michigan sport fish per year, and 19 ppb in 1989 demonstrating little decline within the 7 year interval. For those ingesting <6 pounds of Lake Michigan sport fish per year, the mean serum PCB concentrations were 6.6 ppb in 1982, and 6.8 ppb in 1989. The mean serum PCB concentrations for those consuming <6 pounds of Lake Michigan fish per year are comparable to the mean serum PCB levels of 4 to 8 ppb found in the general population who do not have occupational PCB exposure (Kreiss 1985).

Research has shown that vulnerable communities for exposure to contaminants from fish consumption include Native Americans, minorities, sport anglers, elderly, pregnant women, and fetuses and infants of mothers consuming contaminated Great Lakes fish (Dellinger et al. 1996, Fitzgerald et al. 1996; Lonky et al. 1996; Schantz et al. 1996). These communities may consume more fish than the general population or may have physiologic attributes such as physical and genetic susceptibilities that may cause them to be at greater risk. Higher body burdens of mean serum PCBs and DDE were found in an elderly cohort of Lake Michigan sport fisheaters (i.e., ≥50 years of age) who were compared to nonfisheaters (Schantz et al. 1996). Fisheaters had mean serum PCB levels of 16 ppb while the nonfisheaters had mean levels of 6 ppb. For DDE, fisheaters had mean serum levels of 16 ppb and the nonfisheaters had a mean level of 7 ppb.

In addition, women have been shown to consume Great Lakes sport fish during their reproductive years (Courval et al. 1996, Lonky et al. 1996, Waller et al. 1996). There are also gender differences in fish consumption patterns. A Lake Michigan sport anglers study, with subjects between the ages of 18-34 years, also demonstrated gender differences with males tending to consume more fish than female subjects (Courval et al. 1996). Research has subsequently shown that consumption of contaminated fish by these at-risk populations is associated with adverse human health effects.

3.3.2 Health Effects: Developmental, reproductive, neurobehavioral or neurodevelopmental, and immunologic effects have been reported in studies conducted within the Great Lakes basin and outside the basin. Developmental effects in the form of a decrease in gestational age and low birth weight have been observed in a Lake Michigan Cohort exposed prenatally to PCBs (Fein et al. 1984). These findings have also been observed in offspring of women exposed to PCBs occupationally in the manufacture of capacitors in New York (Taylor et al. 1989).

Reproductive effects have also been reported. Courval and coworkers (1997, 1999) examined couples and found a modest association in males between sport-caught fish consumption with the risk of conception failure after trying for at least 12 months. Studies of New York state anglers have not shown a risk of spontaneous fetal death due to consumption of fish contaminated with PCBs (Mendola et al. 1995) nor an effect on time-to-pregnancy among women in this cohort (Buck et al. 1997).

Neurobehavioral or neurodevelopmental effects have been documented from exposure to persistent toxic substances in newborns, infants, and children of mothers consuming Great Lakes sport fish. Early investigations of the Lake Michigan Maternal Infant Cohort revealed that newborn infants of mothers consuming >6.5 kg/year of Lake Michigan fish had neurobehavioral deficits of depressed reflexes and responsiveness, when compared to non-exposed controls (Jacobson et al. 1984). The fisheating mothers consumed an average of 6.7 kg of Lake Michigan contaminated fish per year, equal to 0.6 kg or 2 to 3 salmon or lake trout meals/month. Prior to study admission, exposed mothers were required to have fish consumption that totaled more than 11.8 kg over a 6-year period. Subsequent studies of the Michigan Cohort have revealed neurodevelopmental deficits in short-term memory at 7 months (Jacobson et al. 1985) and at 4 years of age (Jacobson et al. 1990b), and also growth deficits at 4 years of age associated with prenatal exposure to PCBs (Jacobson et al. 1990a). A more recent investigation of Jacobson's Michigan Cohort has revealed that children most highly exposed prenatally to PCBs showed IQ deficits in late childhood at 11 years of age (Jacobson and Jacobson 1996). Highly exposed children received prenatal PCB exposure equal to at least 1.25 ug/gram (ppm) in maternal milk, 4.7 ng/milliliter (ppb) in cord serum, or 9.7 ng/milliliter (ppb) in maternal serum.

The early findings in newborns by Jacobson have been supported by a more recent study

by Lonky and coworkers (1996) of newborns. In this study, neurobehavioral effects (e.g., abnormal reflexes) were observed in an examination of newborns (12-24 and 25-48 hours after birth), whose mothers had consumed Lake Ontario fish. Newborns, whose mothers had consumed >40 PCB-equivalent pounds of fish in their lifetime, were placed in the high exposure group and were found to manifest a greater number of immature responses and less attention to stimuli. The PCB-equivalent pounds were calculated based upon how the fish was prepared (e.g., extent fat removed) as well as other factors, such as fish species and amount/years of consumption.

In addition to neurotoxic effects observed in newborns, infants, and children, Mergler and coworkers (1997) reported early nervous dysfunction in adults who consumed St. Lawrence River fish. The deficits were found with higher fish consumption. Initial testing for neurotoxic effects were not observed by Schantz and coworkers (1999) in an elderly adult population (i.e., $\geq$50 years) of Lake Michigan sport fisheaters with exposure to PCB and DDE. This study is ongoing.

Immunologic effects have also been reported. Smith's study (1984) demonstrated that maternal serum PCB levels during pregnancy were positively associated with the type of infectious diseases that infants developed during the four months after birth. In addition, incidence of infections has been shown to be associated with the highest fish consumption rate of mothers (i.e., at least three times per month for three years) (Swain 1991, Tryphonas 1995).

Other health effects have been documented with PCB exposure. Elevated serum PCB levels were associated with self-reported diabetes and liver disease in cohorts of Red Cliff and Ojibwa Native Americans (Dellinger et al. 1997, Tarvis et al. 1997). Fischbein and coworkers (1979) found that workers exposed to a variety of PCB Aroclors reported joint pain.

Health effects studies conducted outside the Great Lakes basin have been supportive of the reports from the Great Lakes basin. A summary of these health effects studies can be found in the recent paper published by Johnson and coworkers (1998).

4.0 Societal Responses: Health Advisories for Fish Consumption

The purpose of fish consumption advisories is to protect public health by alerting the residents of potential health risks from consuming contaminated fish (EPA 1995). Advisories can also include information to educate the public about the healthy benefits of fish consumption and to minimize exposure to contaminants in fish by proper preparation and cooking (Tilden et al. 1997). Within the Great Lakes, PCB contamination of Great Lakes fish is generally responsible for health advisories, while mercury contamination is responsible for advisories covering inland bodies of water, such as rivers and lakes (Kamrin and Fischer 1999).

The Great Lakes Sport Fish Advisory Task Force, consisting of environmental and health professionals from the eight Great Lakes states, developed a Health Protective Value (HPV) as a guideline for determining risk from consuming contaminated Great Lakes sport fish (Anderson et al. 1993; Kamrin and Fischer 1999). The HPV is the highest acceptable daily intake of a contaminant (e.g., PCBs) in fish that would not result in a health risk, particularly reproductive and developmental effects, and applies to both sensitive and less sensitive groups (Kamrin and Fischer 1999). For PCBs, the HPV is 0.05 ug PCBs/kg/day. Species of fish are assigned a consumption category that would result in a PCB intake level below the HPV. This value is derived from animal and human study findings, and is similar to the USEPA's reference dose for computing non-cancer risk. There are five consumption categories including *unlimited consumption, one meal a week, one meal a month, one meal every two months,* and *do not eat*. Five of the Great Lakes states have adopted this guideline and two use a version of the HPV. The five include Illinois, Minnesota, Ohio, Pennsylvania, and Wisconsin. Illinois utilizes the HPV for Lake Michigan but also uses the USFDA standard for fish of 2ppm for inland waters. Michigan uses the HPV and the USFDA standard. Indiana also employs the HPV but includes a safety factor for sensitive populations. New York considers acceptable levels (or action levels) of all contaminants in fish and, based on these levels, assigns consumption categories. For example, sensitive populations are permitted to consume only one fish meal a week from water bodies containing fish with PCB levels below the 2 ppm.

Tilden and coworkers (1997) conducted a population-based survey of fish consumption within the eight Great Lakes states. The study results demonstrated that almost 50% of the Great

Lakes fish consumers had an awareness of the health advisories. Of the 50%, approximately 60 % of the males and less than 40% of the females were aware of the advisories. These findings emphasize the importance of targeting health advisories to sensitive groups such as women of reproductive age. The sensitive groups include women of childbearing age and their fetuses and infants, the elderly, sports anglers, and minorities. More information about sensitive groups may be found under the "Weight of Evidence" discussion (3.0).

Studies have shown that having an awareness of health advisories can be successful in changing fishing and fish consumption habits (Fiore et al. 1989; Velicer and Knuth 1994). The communication programs in the Great Lakes generally target white, licensed anglers (Tilden et al. 1997). Written information (i.e., regulation booklets and advisory brochures) is circulated by the government and the fishing industry to licensed anglers, and these sources of information appear to be effective in reducing consumption of contaminated fish. For example, Fitzgerald and coworkers (1999) found that 97% of the men in their study were aware of fish advisories and two-thirds of these men had reduced their fish consumption. This reduction in fish consumption was due to public health intervention strategies such as risk communication along with the use of fish advisories. More recent efforts have been directed toward groups with less awareness of health advisories such as women of childbearing age, minorities, and other frequent fish consumers (Knuth 1995, Tilden et al. 1997)). One of these projects is the ATSDR-funded Consortium of Great Lakes states headed by Dr. Henry Anderson. Anderson and his group have developed outreach materials for women of childbearing age and minority groups which are being utilized by seven of the eight Great Lakes states (Illinois, Indiana, Michigan, Minnesota, New York, Ohio, Wisconsin). These outreach materials such as posters and recipe cards are being adapted by each of the states for their specific needs, and are being distributed at women and childrens' clinics, health fairs, state fairs, and fishing shows to increase health advisory awareness.

Although all of the Great Lakes states have health advisories for contaminants in fish, particularly for PCBs, recent studies demonstrate that sensitive groups may not receive the advisory information (Tilden et al. 1997; Velicer and Knuth 1994). The evaluation of health advisories is an integral part of determining the effectiveness of a program. The USEPA's

Guidance document for fish advisories (EPA 1995) makes recommendations for evaluating the risk communication efforts for fish advisories and provides a step-by-step approach for conducting an evaluation of an existing program. Program evaluation is necessary to determine 1) if the health advisory is reaching the target population, 2) if it is being implemented properly, 3) if it is effective, 4) the cost, and 5) the cost relative to effectiveness (Windsor et al. 1994).

5.0 ATSDR's Program: a Model for the Great Lakes Water Quality Agreement

The ATSDR's Great Lakes Human Health Effects Research Program (GLHHRP) serves as a model by which the requirements of the human health component of the Great Lakes Water Quality Agreement are being met. The goals of the GLHHRP are to 1) identify the populations at risk who may be exposed to chemical contaminants from the Great Lakes, and 2) prevent the potential adverse human health effects that research has demonstrated is associated with exposure. These goals represent the program's public health focus intended to protect the health of populations consuming contaminated Great Lakes fish. ATSDR has established an applied research strategy to achieve these goals based upon the traditional model of disease prevention (De Rosa and Johnson 1990; Johnson et al. 1998). These strategies are key requirements of the human health component of the Great Lakes Water Quality Agreement.

The GLWQA calls for the LaMPs ". . . to include a definition [description] of the threat to human health . . . posed by Critical Pollutants, singly or in synergistic or additive combinations . . . ." (IJC 1994). The GLWQA also calls for the establishment of a surveillance and monitoring system, one of whose purposes it to identify emerging problems. For ATSDR, identification has involved identifying vulnerable populations and cohorts of populations who consume contaminated fish and have a potential for developing adverse human health effects (Anderson et al. 1996; Courval et al. 1996, 1999; Daly et al. 1996; Fitzgerald et al. 1996, 1999; Schantz et al. 1996; Stewart et al. 1999; Vena et al. 1996; Waller et al. 1996). The ATSDR cohort populations are part of a surveillance and monitoring system to identify emerging problems of long-term health effects associated with consumption of contaminants in fish.

Evaluation is another ATSDR strategy element used to determine causal linkages or conclusions regarding biologic plausibility. Early reports from the ATSDR's GLHHRP have demonstrated exposure associations between consumption of contaminants in Great Lakes fish and body burdens particularly for those with high fish consumption. The program has entered into a second evaluation phase in which associations are being established between body burdens of contaminants (e.g., in serum) and health effects observed in humans and animals.

As with the GLWQA, implementation is an integral part of ATSDR's strategy. Having helped to establish the pathway of exposure for at-risk populations, ATSDR's prevention strategy involves risk communication and health education to minimize the public's exposure to contaminants in fish (Tilden et al. 1997). Health advisories for fish consumption are important means of communicating to the public the potential toxic effect from contaminants in Great Lakes fish. An ATSDR-funded research group has helped to develop uniform health advisory guidelines for fish consumption that is being utilized by the Great Lakes states. In addition to the funded research, ATSDR is presently preparing a report assessing health advisories for fish consumption within the Great Lakes states. This report will include an examination of some of the outreach approaches (e.g., pamphlets, posters, Internet) used by the Great Lakes states to disseminate health advisory information. As a further component of the prevention strategy, ATSDR has an ongoing program dealing with the effect of mixtures of chemicals found in the Great Lakes and other sites to determine synergistic or additive effects of these chemical mixtures (Hansen et al. 1998). Within the next year, a toxicological profile will be published by ATSDR describing the state-of-the-science for chemical mixtures found in the Great Lakes and other hazardous waste sites.

As part of the impact assessment, ATSDR has established a process by which the GLHHRP projects are reviewed. Results of these research projects are customarily published to expand the public's awareness of potential adverse human health effects from consuming contaminated fish. ATSDR has also been participating in the Lakes Erie, Michigan, and Superior LaMP work groups, and has utilized this opportunity to 1) develop a human health section document that can be utilized as a prototype for all LaMPs, 2) inform the governmental and non-governmental agencies and the public about recent findings from the ATSDR funded research,

and 3) develop an awareness about the current health-related issues in the Great Lakes basin that can assist in the direction of the GLHHRP.

This strategy and its component elements have represented major strides in helping to fulfill the requirements of the Great Lakes Water Quality Agreement by delineating the potential human health threat from contaminants in Great Lakes fish and by implementing actions that will protect human health. Having achieved these major steps, ATSDR is now making an effort to advance the science in relatively pristine areas such as health effects from multiple chemicals found in the Great Lakes and other sites (Hansen et al. 1998), and the development of biomarkers of exposure. This step-by-step process will also be instrumental in building a data base of knowledge that can be utilized in dealing with other health and environmental related issues both nationally and internationally.

6.0 Conclusions and Recommendations

Much has been accomplished. Much still needs to be done. Within the ecosystem, there are encouraging signs and successes. For example, contaminant declines have been observed at most Great Lakes sites as evidenced in the eggs of herring gulls (Environment Canada and EPA 1999). In southern Lake Michigan during 1998, yellow perch seemed to be spawning successfully and lake sturgeons have been reported to reproduce successfully in three tributaries of Lake Michigan's Green Bay.

In the human health sector, research has identified fish consumption as the major pathway of exposure to some contaminants from Lake Michigan and other Great Lakes. Body burdens from consumption of contaminated fish have been noted in vulnerable communities. Human health effects have subsequently been observed. Despite these compelling findings, issues related to environmental exposures and human health still remain. Health research needs to continue, but a shift in priorities is now needed to prevention, intervention, and collaborative activities. In addition, work still needs to be done in these areas. Recommendations are being made for:

- biologic markers of exposure, effect, and susceptibility
- health effects of chemical mixtures

- endocrine disruptors
- PCB congeners and their health effects
- health advisories and outreach approaches
- surveillance and monitoring programs

6.1 Biologic Markers of Exposure Effect and Susceptibility

Research has demonstrated that regular high levels of fish consumption can result in high body burdens of lipophilic contaminants such as PCBs and that the body burdens of these contaminants remains relatively constant in the body even after exposure cessation. For the goal of prevention, improved markers are needed to indicate biologic changes that predict health impairment or disease (NRC 1989) and the preclinical signs of disease (De Rosa and Johnson 1996). The three categories of biologic markers include markers of exposure, effect, and susceptibility (NRC 1989). A biologic marker of exposure may be the body burden, indicative of recent or past exposure. A marker of effect may include health impairment (e.g., altered function of the luteinizing hormone for ovarian function) or overt disease (e.g., fertility problems). A pre-existing condition affecting the dose of a substance to the target tissue is an example of a biologic marker of susceptibility. The ATSDR-funded research and other research projects are examining body burdens of contaminants in serum, reproductive problems related to conception, and other health-related problems, that will be instrumental in identifying early warning signs requiring intervention. However, the biologic markers of exposure and effect often lack the precision to identify those who have an exposure, impairment, or disease, and those who do not. For this reason, additional research is needed to develop biologic markers that clearly identify the concentration of contaminants and the point in the human physiological process beyond which lasting adverse health effects will be observed.

6.2 Health Effect of Chemical Mixtures

Within our present state-of-knowledge, the human research demonstrating health effects from consumption of contaminated fish can be said to be relatively sound. Although these studies, in most cases, have made associations between a single contaminant detected in fish and

body burden or health effects, detections of multiple chemicals have been found in Great Lakes fish (Humphrey 1988; Dellinger et al. 1996). Our present state-of-knowledge is vastly limited in identifying the subtle effect of multiple chemicals detected, even at low levels, in contaminated fish. For this reason, research is needed to clearly delineate whether a synergistic or additive health effect occurs with multiple chemicals, and with a combination of chemicals having similar properties.

6.3 Endocrine Disruptors

Research has demonstrated that many of the contaminants found in fish from Lake Michigan and other Great Lakes have been shown to adversely affect the endocrine system of animals and humans. An environmental endocrine disruptor is ". . . an exogenous agent that interferes with the synthesis, secretion, transport, binding, action, or elimination of natural hormones in the body that are responsible for the maintenance of homeostasis, reproduction, development, and/or behavior" (EPA 1997a). Some of the known endocrine disrupting chemicals include atrazine, chlordanes, DDT and metabolites, dieldrin, dioxins and furans, PCBs, and toxaphene (EPA 1997b). These are contaminants that have been detected in Lake Michigan and other Great Lakes. Other substances, detected in the Great Lakes, are considered probable endocrine disruptors. These include cadmium, hexachlorobenzene, lead, mercury, and mirex. Although research continues on reproductive (Buck et al. 1999, Courval et al. 1999) and other effects that may be associated with exposure to endocrine disrupting chemicals, our knowledge about these substances in humans remains limited. Epidemiologic research needs to quantify the magnitude of exposures and effects of substances considered to be endocrine disruptors (EPA 1997a). Since endocrine disrupting chemicals, such as PCBs and DDT, have been detected simultaneously in fish, their effect as chemical mixtures also requires investigation.

6.4 PCB Congeners and Their Health Effects

Further human research is needed to identify the specific PCB congeners associated with adverse human health effects. The use of the capillary column gas chromatography, starting in

the late 1980s and early 1990s, has enabled laboratories to identify the 209 PCB congeners (Communication with Virlyn Burse 1/25/00). Stewart and coworkers (1999) found that the most heavily chlorinated PCB homologues (i.e., 7 or 8 chorines per PCB biphenyl ring) were significantly higher in the fetal cord blood of infants whose mothers had consumed Lake Ontario fish. These highly chlorinated and persistent PCB homologues were also detected in fish from Lake Ontario. Animal studies have supported this observation that highly chlorinated PCBs are responsible for adverse health effects. Congener-specific studies will help to identify those congeners that are most likely to adversely influence human health and require public health intervention.

6.5 Health Advisories and Outreach Approaches

ATSDR-funded research has examined health advisories for fish consumption within the Great Lakes (Tilden et al 1997). The study findings demonstrated that approximately 50% of survey respondents were aware of health advisories with awareness being lowest in women. Studies have shown that sport fish consumption advisories and outreach information can minimize exposures to contaminants from sport fish consumption. Information about fish advisories has been successfully disseminated to licensed anglers through the recreational fishing industry and governmental offices, but has not been effective for non-licensed anglers and women who are among the at-risk populations. Added effort needs to be directed towards informing and educating at-risk populations, and should include the selection of outreach material shown to be effective in modifying behavior (e.g., proper fish preparation). In addition, a single uniform fish advisory guideline is needed that can easily be adapted to specific state needs. The work of the Great Lakes Sport Fish Advisory Task Force can be used as a template for developing comprehensive guidelines, that will fulfill the needs of all states, not just the Great Lakes states. Factors to be considered include the size of an average fish meal, reduction in contaminant levels due to proper preparation and cooking, and other contaminants (aside from PCBs) in fish (Kamrin and Fischer 1999). ATSDR and ATSDR-funded research are continuing to examine this important area.

6.6 Surveillance and Monitoring Programs

Surveillance and monitoring programs have been successful in identifying subtle health effects associated with body burdens of contaminants associated with consumption of Great Lakes fish. ATSDR has participated in the Lake Michigan Tributary Monitoring Project, providing information about surveillance and monitoring of cohorts in the ATSDR-funded research. Input on health surveillance and monitoring should continue to be an integral part of the Lake Michigan Monitoring Project and other monitoring projects concerned with environmental issues and health.

Although progress has been made in defining the health threat from Great Lakes pollutants, important issues remain requiring our diligent effort. The Great Lakes Water Quality Agreement, under the Research and Development annex, calls for ". . . develop[ing] approaches to population-based studies to determine the long-term, low-level effects of toxic substances on human health" (IJC 1994), For the public health arena, there are a number of issues that will help to identify these long-term, low-level health effects, as discussed in the previous paragraphs. Research in these areas will provide a more comprehensive view of the threat to human health from critical pollutants, and enable ATSDR and other public health agencies to utilize this knowledge to protect the public health more effectively. In addition, a shift in priorities is now needed to prevention, intervention, and collaborative activities. These steps will also be critical to environmental agencies setting guidelines and standards to protect the Great Lakes populations and our entire nation.

Internet Information Resources and Further Reading Lake Michigan Human Health Issues

A. General Internet Resources and Readings

US Environmental Protection Home Page
http://www.epa.gov/

USEPA Great Lakes National Program Office
http://www.epa.gov/glnpo

USEPA Region 5
http://www.epa.gov/

U.S. Centers for Disease Control and Prevention
http://www.cdc.gov/

U.S. Agency for Toxic Substances Disease Registry
http://www.atsdr.cdc.gov/

U.S. ATSDR Great Lakes Health Effects Program
http://www.atsdr.cdc.goc/grlakes.html

States

Illinois Department of Health

Indiana Department of Health

Michigan Department of Community Health
http://www.mdch.state.mi.us/

Wisconsin Department of Health
http://www.dhfs.state.wi.us/

Readings

ATSDR (Agency for Toxic Substances and Disease Registry). 1993. Aldrin/Dieldrin Fact Sheet. Atlanta, Georgia: U.S. Department of Health and Human Services.

ATSDR (Agency for Toxic Substances and Disease Registry). 1995. Chlordane Fact Sheet. Atlanta, Georgia: U.S. Department of Health and Human Services.

ATSDR (Agency for Toxic Substances and Disease Registry). 1999. Chlorinated Dibenzo-p-Dioxins Fact Sheet. Atlanta, Georgia: U.S. Department of Health and Human Services.

ATSDR (Agency for Toxic Substances and Disease Registry). 1995. DDT, DDE, and DDD Fact Sheet. Atlanta, Georgia: U.S. Department of Health and Human Services.

ATSDR (Agency for Toxic Substances and Disease Registry). 1997. Hexachlorobenzene Fact Sheet. Atlanta, Georgia: U.S. Department of Health and Human Services.

ATSDR (Agency for Toxic Substances and Disease Registry). 1999. Mercury Fact Sheet. Atlanta, Georgia U.S. Department of Health and Human Services.

ATSDR (Agency for Toxic Substances and Disease Registry). 1997. Polychlorinated Biphenyls Fact Sheet. Atlanta, Georgia: U.S. Department of Health and Human Services.

ATSDR (Agency for Toxic Substances and Disease Registry). 1998. Polychlorinated Biphenyls Toxicological Profile (updated draft). Atlanta, Georgia: U.S. Department of Health and Human Services.

ATSDR (Agency for Toxic Substances and Disease Registry). 1997. Toxaphene Fact Sheet. Atlanta, Georgia: U.S. Department of Health and Human Services.

International Joint Commission. Revised Great Lakes Water Quality Agreement of 1978 as Amended by Protocol Signed November 18, 1987. Reprint February, 1994.

U.S. EPA and Government of Canada, 1995. The Great Lakes: An Environmental Atlas and Resource Book.

Johnson, B.L., H.E. Hicks, D.E. Jones, W. Cibulas, A. Wargo and C. T. De Rosa. 1998. Public Health Implications on Persistent Toxic Substances in the Great Lakes and St. Lawrence Basins. Journal of Great Lakes Research. 24(2): 698-722.

B. Internet Resources and Further Readings for Air:

EPA Office of Air and Radiation
http://www.epa.gov/oar/oarhome.html

U.S. EPA Health Effects Notebook for Hazardous Air Pollutants
http://www.epa.gov/ttn/uatw/hapindex.html

OSHA Indoor Air page:
http://www.osha-slc.gov/SLTC/indoorairquality/index.html

C. Internet Resources and Further Readings for Drinking Water:

U.S. EPA Office of Ground Water and Drinking Water Home Page
http://www.epa.gov/safewater/about.html
http://www.epa.gov/OGWDW/wot/appa.html
http://www.epa.gov/ogwdwooo/hfacts.html

U.S. EPA, How Safe is my Drinking Water? Office of Ground Water and Drinking Water
http://www.epa.gov/OGWDW/wot/howsafe.html

U.S. EPA, Current Drinking Water Standards - National Primary and Secondary Drinking Water Regulations. Office of Groundwater and Drinking Water web site at
http://www.epa.gov/OGWDW/wot/appa.html

U.S. EPA, Consumer Confidence Reports. Fact Sheet. At web site
http://www.epa.gov/safewater/ccr/ccrfact.html

USFDA Food borne Pathogenic Microorganisms and Natural Toxins Handbook Web Page
http://vm.cfsan.fda.gov/~mow/chap24.html

US Center for Disease Control. Cryptosporidiosis Fact Sheet.
http://www.cdc.gov/ncidod/diseases/crypto/cryptos.htm

Readings

Wisconsin Department of Natural Resources, 1998. "Cryptosporidium: A Risk to our Drinking Water." Fact Sheet. Available on WDNR web site at
http://www.dnr.state.wi.us/org/water/dwg/Crypto.htm#what steps Revised June 1, 1998.

D. Internet Resources and Further Readings for Recreational Water

U.S. EPA, Office of Water, EPA's BEACH Watch Program, 1999 Update
http://www.epa.gov/OST/beaches/update.html

U.S. EPA's BEACH Watch Program Homepage
http://www.epa.gov/OST/beaches/

U.S. EPA Office of Water, BEACH Watch Program Homepage.
http://www.epa.gov/OST/beaches/

U.S. EPA Office of Water, BEACH Watch Program. Local Beach Health Information.
http://www.epa.gov/OST/beaches/local/

Natural Resources Defense Council (NRDC). Testing the Waters - 1999 - A Guide to Water Quality at Vacation Beaches
http://www.igc.org/nrdc/nrdcpro/ttw/titinx.html

U.S. EPA's BEACH Watch Program Homepage at http://www.epa.gov/OST/beaches/
Natural Resources Defense Council (NRDC). Testing the Waters - 1999 - A Guide to Water Quality at Vacation Beaches. at http://www.igc.org/nrdc/nrdcpro/ttw/titinx.html

Corbett, S.J., Rubin, G.L., Curry, G.K., Kleinbaum, D.G. & the Sydney Beach Users Study Advisory Group 1993, 'The health effects of swimming at Sydney beaches', American Journal of Public Health, vol.83, no.12, pp.1701-06 .
URL: http://www.epa.nsw.gov.au/soe/95/14_2s2.htm

E. Internet Resources and Further Readings for Fish/Food Consumption

U.S. EPA Fish Consumption Advisory Information
http://www.epa.gov/OST/fish/

States

Illinois Department of Natural Resources.
Indiana Department of Natural Resources. Indiana Fish Advisory

Michigan Department of Community Health. Michigan Fish Advisory
http://www.mdch.state.mi.us/pha/fish/index.htm

Wisconsin Department of Natural Resources. Wisconsin Fish Advisory
http://www.dnr.state.wi.us/org/water/fhp/fish/advisories/

Illinois Fish Advisory

F. Internet Resources and Further Readings for Health Effects Information

ATSDR's Toxicological Profiles
http://www.atsdr.cdc.gov/toxpro2.html

ATSDR HAZDAT Database: Hazardous Materials and their Human Health Effects
http://atsdr1.atsdr.cdc.gov:8080/hazdat.html

ATSDR, Public Health Implications of Exposure to Polychlorinated Biphenyls (PCBs)
http://www.atsdr.cdc.gov/DT/pcb007.html

U.S. EPA Mercury Study Report to Congress
http://www.epa.gov/ttn/oarpg/t3/reports/volume5.pdf